Mitochondrial Defects and Their Role in Development of Prostate Cancer

Presenting author: Phd student/laboratory assistant Liana RamiShvili[a]

Coauthor: Invited lecturer, Dr. of Biology Manana Alibegashvili[a]

E.mail: liana.ramishvili@tsu.ge

[a] Division of Cellular and Molecular Biology, Department of Biology,
Faculty of Exact and Natural Sciences, Iv. Javakhishvili Tbilisi State University;
13 University str.

One of the characteristic changes of tumor formation is accumulation of genetic disorders in mitochondrial and nuclear genome. Mitochondrial disorders, from its side, are responsible for failure of metabolism, apoptosis, cell growth, formation of reactive oxygen species, etc. Overprpoduction of reactive oxygen species (ROS) significantly impacts the respiration chain enzymes and entirely the antioxidant system of mitochondria. Finally this may become a favorable condition for normal cells transformation.

The purpose of the presented work was to study the mitochondrial defects and to establish their role in prostate cancer development.

Experimental results demonstrate significant increase of the activity of mitochondrial succinate dehydrogenaze (complex II) of the malignant epithelial cells of prostate, and slight changes in cytochrome oxydase (complex IV) activity. Also significant activation of the antioxidant system (glutathione-dependant system) of mitochondria in prostate malignant epithelial cells was revealed.

The above mentioned mitochondrial changes (II and IV complexes of respiration chain, activity of the antioxidant system) partially demonstrate the alterations in mitochondrial energy metabolism, which from its side, may indicate to resistance of prostate cancer cells and correspondingly to intensification of proliferation processes.